

Date: February 10, 2004

Subject: Human Health Risk Assessment for the Diamond Lake Restoration Project—Toxic Algae Blooms

To: Sherri Chambers, IDT leader

Introduction

The Umpqua National Forest, in cooperation with multiple state and federal agencies, proposes to use two formulations of the fish toxicant, rotenone, to eradicate unwanted tui chub fish in Diamond Lake. This action is proposed in order to improve both water quality and the trout fishery which have been substantially diminished due to the tui chub population. These rotenone formulations would be applied under two of the four alternatives assessed in the Diamond Lake Restoration Environmental Impact Statement (EIS). This report addresses the risks to human health associated with toxic algae blooms related to the four Alternatives presented in Chapter 2 of the EIS. The risks to human health associated with the use of rotenone are analyzed in a separate report for this EIS (Obery and Fontaine, 2004).

AFFECTED ENVIRONMENT

Algal Blooms and Human Health

Cyanobacterial algal blooms occurred in Diamond Lake during the summers of 2001-2003. The hazardous blooms are the likely result of optimal climatic conditions in conjunction with the ecological changes in the lake due to the large populations of tui chub. The currently large population of tui chub has the potential to enhance the detrimental algal blooms in two ways. First, tui chub can eat the larger-sized zooplankton reducing their populations to the point that they no longer effectively graze on the algal cells and thus no longer keep the algal population in check. Secondly, the tui chub population can increase the nutrient concentration in the lake through excretion of nitrogen and phosphorus in forms available for algal growth, essentially adding fertilizer for the plant population to expand.

Cyanobacteria, also known as blue-green algae, are single-celled aquatic organisms found in surface waters worldwide. Given the recent ideal conditions for plant growth in Diamond Lake, vast blooms have occurred during the summer months. The resulting high cyanobacterial algal concentrations can produce toxins that have been implicated in human health problems ranging from skin irritation and gastrointestinal upset, to death from liver or respiratory failure (Chorus and Bartram 1999; Chorus 2001)

The two main species of toxin-producing cyanobacteria associated with the blooms in Diamond Lake are *Anabaena flos-aquae* and *Microcystis aeruginosa*, with *Anabaena* more prominent (Eilers and Kann 2002). *Anabaena flos-aquae* is most frequently

associated with the powerful neurotoxin anatoxin-a; however, *A. flos-aquae* can also produce hepatotoxic microcystins. *Microcystis aeruginosa* produces microcystins (Yoo et al. 1995). These species, like all cyanobacteria, also have compounds in their cell walls which are probably responsible for the adverse skin, eye, mucosal, and digestive reactions reported by people who have come in contact with them (Chorus 2001). The amount of toxin produced by cyanobacteria is highly variable but is generally related to their density or biomass in the water column (Chorus and Bartram 1999). Moreover, because cyanobacterial cells are buoyant and frequently concentrate at the surface, wind may concentrate cells along the shoreline or in bays, causing toxins to be at sufficiently high levels, and sufficiently accessible, to be dangerous to humans (as well as other animals) who come in contact with them (Chorus and Bartram 1999).

Anatoxin-a

Anatoxin-a is an alkaloid that acts as a post-synaptic depolarizing neuromuscular blocking agent, causing nerve impulses to over-stimulate muscle cells (particularly those involved in breathing), leading to convulsions and death by asphyxiation (Carmichael 1994). Animals exposed to anatoxin-a through drinking *Anabaena*-contaminated water, grooming scum from their coats or feathers, or in laboratory tests, die of the poison within minutes or hours. Cattle, pet dogs, waterfowl and others have met their deaths in this way (Backer 2002).

In 2002 the first human death presumed due to *Anabaena* toxin occurred near Madison, Wisconsin, when a teenage boy died 48 hours after diving and swimming in a scum-coated pond. He was later reported to have swallowed at least one mouthful of pond water, suffering nausea and diarrhea, then seizures, before dying. However, because of the length of time between exposure and death, some uncertainty remains as to whether anatoxin was responsible, but no other possible explanations have been found. Several friends who went into the pond at the same time also developed gastrointestinal symptoms, but only one, who had also gone completely underwater, became acutely ill with diarrhea (Milwaukee Journal Sentinel, 2002).

It has been assumed that humans have suffered less than birds and other animals from anatoxin poisoning because the blooms make the affected water so disagreeable that people voluntarily limit their exposure. Nonetheless, although few people would be likely to actively drink from such a source, the possibility of recreational exposure through accidental immersion and ingestion or aspiration exists, especially if boating activities are going on. Even a scummy lake can look irresistible on a blazing afternoon, and if mats of bloom wash up onto the shoreline, there may be considerable risk to investigative toddlers (Chorus 2001). Moreover, at present, insufficient data exists to establish reliable safety thresholds for human exposure. Human studies are lacking and although there is experimental laboratory data generated from mice, it is considered inadequate for the formulation of human Tolerable Daily Intake standards, whether for toddlers or adults (Chorus and Bartram 1999). It is also difficult to know how much toxin is actually out there at any given time. Tests to determine toxin levels take time; the bloom may be in a period of exponential growth, with toxin levels changing fast, or be dying off, in which case breakdown of the cells may be releasing large amounts of toxin into water that looks free of problems. Wind and wave motion may concentrate the bloom and its toxins in one part of the lake, leaving other areas apparently free of problems. In addition, uncertainty exists as to accumulation of anatoxin-a in fish tissue, so that fishermen

may be at risk in eating their catches (Falconer 1993). Finally, although it does not appear to be the case that recurrent low exposure to anatoxin-a leads to health problems later on, this is not established, and there is concern that people so exposed may become sensitized and develop increasingly more severe reactions with each new exposure (Backer 2002).

Microcystins

Microcystins were first isolated from *Microcystis aeruginosa* but are also produced by other species, including *Anabaena*. These hepatotoxins (liver toxins) are powerful cyclical peptides which disrupt the structure of liver cells, causing cell destruction, liver hemorrhage, liver necrosis, and death (Carmichael, 1994).

Microcystin toxins in the water supply of a renal dialysis clinic in Brazil resulted in the illness of 110 of 113 patients, including the deaths of 55 patients from liver hemorrhage and liver failure (Backer 2002). Microcystin delivered by the nasal route to experimental rodents also resulted in erosion of the nasal mucosa to the point of hemorrhage. In addition to hepatotoxicity, long-term laboratory animal studies indicate that microcystins act as liver tumor promoters and teratogens (Falconer et al. 1988). Anecdotal evidence for such chronic effects on humans is based in large part on the high rates of primary liver cancer in the rural regions of China where drinking water was obtained from ditches and ponds with large cyanobacteria loads. Where drinking water supplies have been changed from surface sources to deep wells, cancer rates have begun to drop (although other contributing factors are also being addressed). Other studies have shown microcystins to promote liver tumor development in lab animals, and the survivors from the Brazilian dialysis clinic are being monitored for long-term effects, including development of liver cancers (Chorus and Bartram, 1999).

Microcystin poisoning has been implicated in the largest number of cyanobacteria-associated animal deaths worldwide, and enough work has been done, both with rodents and pigs, on microcystin effects at various levels of exposure, that the World Health Organization (WHO) has issued a provisional guideline of 1 ug/L for microcystin concentration in drinking water. With actual microcystin concentration data frequently unavailable, alert level guidelines based on cell counts have been established for *Microcystis* (as well as other cyanobacteria) blooms in drinking and recreational waters (Yoo et al. 1995; Chorus and Bartram 1999). Alert Level 1 is 500 cells/ml and requires increased frequency of monitoring; Alert Level 2 is 2000 cells/ml and requires alerting the public and posting water bodies; Alert Level 3 is 15,000 cells per ml and warrants a recreational closure for water contact recreation. Alert level 3 was shown in an epidemiological study to result in skin and mucosal irritation and gastrointestinal symptoms.

Microcystin toxins can be released into the water, remaining even after the *Microcystis* bloom has visually dissipated either from natural senescence or treatment by algaecides (e.g., copper sulfate); thus, caution should be exercised for drinking and recreational water sources even when no scum or cells may be visible (Lam, et al. 1995).

Toxicity Studies

Acute toxicity of the *Anabaena flos-aquae* toxin anatoxin-a has been studied using mice; administration of the toxin has been by intravenous, intraperitoneal, nasal, and oral routes. LD50 (lethal dose, at which 50% of the mice died) for intravenous administration is less than 100 ug/kg of body weight (Fawell and James 1994, cited in Chorus and Bartram 1999); for intraperitoneal it is 375 ug/kg body weight; nasal administration average lethal dose is 2000 ug/kg of body weight and 5000+ ug/kg body weight was the average lethal dose for anatoxin-a given orally (Fitzgeorge, et al. 1994, cited in Chorus and Bartram 1999).

Subacute toxicity studies in which 51 or 510 ug/kg body weight was given to mice in drinking water, or 16 ug/kg given to mice intraperitoneally for 21 days, resulted in no deaths and no change in weight or body chemistry of the mice (Fawell and James 1994, cited in Chorus and Bartram 1999). Intraperitoneal administration of anatoxin-a to pregnant hamsters at 200 ug/kg of body weight daily, or 125 ug/kg 3 times a day during days 6-15 of their pregnancy resulted in no toxic symptoms to the mothers, but nearly all fetuses showed stunted growth; those in the three times daily group also showed hydrocephaly (Astrachan et al. 1980). Pregnant mice fed anatoxin-a hydrochloride at 3000 ug/kg body weight during days 6-15 had litters with slightly lower fetal weight than untreated mothers (Fawell and James 1994, cited in Chorus and Bartram 1999).

Acute *Microcystis* toxicity has mostly been studied using microcystin-LR, which is rapidly transported to the liver when administered intraperitoneally or intravenously (Falconer et al. 1986; Runnegar et al. 1986; and others, cited in Chorus and Bartram 1999) with LD50 ranging from 25-150 ug/kg body weight (the generally accepted value is 50-60 ug/kg); average lethal dose for oral administration is 5000 ug/kg body weight in one mouse strain (Fawell, et al., 1994) but 10,900 ug/kg in a different strain (Yoshida et al. 1997). Microcystin-LR is not broken down by stomach enzymes, but absorbed through the intestines; a difference has been seen between young mice (5 weeks old) and older mice (32 weeks old) given oral doses of 500 ug/kg of body weight, in which only the older mice suffered apparent liver damage. Upon examination of control mice of both ages, the older mice were found to have age-related changes to the surface cells of the stomach lining and intestine that may result in greater absorption and toxic effects in older mice (Ito et al. 1997a, cited in Chorus and Bartram 1999).

Nasal exposure does extensive damage to the nasal tissues and the average lethal dose is comparable to that of intraperitoneal administration. Liver damage, as well as nasal damage, is seen. Subacute dosage (31 ug/kg) administered over several days resulted in nearly as much damage to the liver as the acute dosage of 500 ug/kg body weight.

Long-term subacute exposure in both mouse and pig studies shows liver damage occurring to mice given pure microcystin-LR as well as to both mice and pigs given extracts of *Microcystis aeruginosa* in their drinking water for up to one year. Based on the results of the pig study, a lowest observed adverse effect level (LOAEL) value of 100 ug/kg body weight per day for microcystin-LR equivalents was developed (Falconer et al. 1994, cited in Chorus and Bartram 1999).

Effects on pregnant mice and their fetuses were studied; nine mother mice in the highest dosage group (2000 ug/kg body weight during days 6-15 of pregnancy) died during the dosing period, and fetuses at this level were behind in weight and skeletal ossification (Fawell et al. 1994, cited in Chorus and Bartram 1999).

Studies to determine carcinogenic effects from microcystins show liver nodules developing in mice given intraperitoneal microcystin-LR at 20 ug/kg body weight 100 times in a 28-week period; the nodules persisted after dosing was ended. Oral administration during the same period at 80 ug/kg did not result in liver injury or liver nodule formation. However, microcystin acts to inhibit protein phosphatases, and substances which do so are considered to be tumor promoters (Ito et al. 1997b).

Based upon the studies done with microcystin-LR, the World Health Organization has established a provisional drinking water standard of 1 ug/kg body weight of microcystins in 2 L of drinking water consumed daily (Chorus and Bartram 1999).

Cyanotoxin Risks in Recreational Settings

Treatments such as ozonation can be used to purify drinking water supplies contaminated with cyanobacteria, but it is unlikely to be able to treat whole lakes and other recreational waters this way (Chorus 2001). And while not all cyanobacteria, and perhaps not all cyanobacterial blooms, are toxic, precaution is the best policy (Yoo, 1995). A study of eight lakes in Alberta found microcystin toxins present in 37 of 39 samples taken (Kotak et al 1993); an epidemiological study in Australia found health problems including skin, eye and mucosal irritation and gastrointestinal complaints, even at relatively low levels of cyanobacterial presence: 20,000 cells/mL, or about enough to discolor the water (Pilotto et al. 1997, cited in Chorus and Bartram 1999).

For accumulations of cyanobacteria in recreational waters, monitoring of cell density levels and informing users of the risks or restricting access are the only immediate options. Cell densities of 15,000/mL may contain enough cyanotoxin to cause problems for especially sensitive or allergic people. If *Microcystis aeruginosa* is present, microcystins at subacute levels may be ingested or inhaled by swimmers, skiers, windsurfers or kayakers. Cell densities of 100,000 and higher are considered likely to produce microcystin concentrations of 20 times the WHO guidelines for drinking water, at which point even boaters are considered at risk. Scum formation is likely at this density, as the concentration of buoyant cells rises and covers the surface.

This buoyancy creates a monitoring problem, since light winds or other disturbances of the water can concentrate the cells to even greater densities in some areas while causing other areas to appear bloom-free. Dense mats of bloom pushed near or onto the shore may have up to a thousand fold increase in toxin concentration over what monitoring in another area, or the same area a few hours earlier, has indicated (Chorus and Bartram 1999). Pets, wading children, and others engaged in near-shore activities may be at high risk in these areas (Watanabe, et al. 1996). Accidental ingestion of this concentrated scum could cause serious health problems, making close

and frequent monitoring at multiple sites and, if necessary, restriction of access, a matter of urgent importance (Chorus and Bartram 1999).

Table 1. Summary of the toxicity information available on algae toxins.

	Anatoxin-a	Microcystin
Proposed Safe Concentrations	Not established— 100ug/l would likely kill a dog drinking from the lake (Carmichael),	1 ug/kg/day (Proposed by World Health Organization)
Levels observed in D. Lake	Up to 300ug/L	Up to 2.54 ug/L
Non-accute effects	unknown	Probably carcinogenic

ENVIRONMENTAL EFFECTS

Predicting the effect of tui chub removal on the magnitude and annual trends of toxic algal blooms in Diamond Lake is difficult due to uncertainty in such factors as inter-annual climatic variability, restocking of the lake with rainbow trout, internal nutrient recycling from the sediments, and response of the zooplankton and benthic communities. Nonetheless, it is clear that based upon paleolimnological data that the lake began changing shortly after being stocked with trout, and that the greatest increases in *Anabaena* were associated with increased populations of tui chub. It is likely then that under current conditions of extremely high numbers of tui chub, that toxic *Anabaena* blooms (and subsequent lake closures) will continue annually, with the severity determined by inter-annual variability in climate. In other words, under current high nutrient excretion rates by tui chub, the main determinant of inter-annual toxic bloom variability will be the recurrence interval of the calm, sunny, warm conditions that tend to favor blooms of cyanobacteria. However, under a given set of climatic conditions the likelihood of large cyanobacterial blooms would be diminished (although periodic blooms can still be expected) as available nutrients decrease in the water column with chub removal.

Direct Effects:

Direct effects to human health would be those that occur in the short-term, over a period of several years, and at the immediate site of Diamond Lake itself.

Under Alternative 1, the general visiting public would continue to be at risk of exposure to lake waters contaminated with blue-green algae toxins. Elevated concentrations of either anatoxin-a or microcystins, or both, are predicted to continue during warm, sunny weather as long as tui chub remain abundant in Diamond Lake. The likelihood of exposure would be lessened by annual water monitoring for the algae toxins that would be used to alert lake users and to trigger necessary lake closures.

However, as time goes on, the chances of public exposure by accident or by ignoring the warnings and closures would increase simply due to the long-term presence of the risk coupled with the popularity of Diamond Lake to the recreating public. Since the concentrations of toxins can be variable in the lake depending on location, depth, shoreline configurations, wind effects and rapid algal population shifts, the toxicity monitoring may not catch hot spots of concentrated toxins and timely lake closures may not always occur. In this event, swimmers or boaters could potentially receive a dose of the toxin(s) and become seriously ill. If such a dermally exposed person were to accidentally swallow concentrated toxins at the same time, there may be a potential of death, such as occurred in Wisconsin in 2002. However, the potential for mortality remains extremely low under any of the alternatives.

Agency employees or private contractors involved in the toxicity monitoring would also be at continued risk under Alternative 1. Since these administrative workers would be seeking out areas of concentrated toxins, the consequences of accidental submersion would be relatively high. Such an accident could result in the direct effect of serious illness or even death if some of the toxin is swallowed during the accident. These potential direct effects to the health of administrative personnel would be lessened by safety training and the implementation of pre-planned mitigation such as washing with clean water immediately following an incident.

Under Alternative 1, the domestic water from both the shallow and deep aquifer wells would not be at risk of contamination from algal toxins. This is because the toxins are expected to be filtered out of the water by the sediments and rock that exists in the ground water environment (Chorus and Bartram, 1999).

Under either Alternative 2 or 3, prior to the rotenone application when the lake would be drawn down by about 8 feet in elevation, toxic algae blooms could be significant if weather conditions were conducive to blooms. Moreover, wave action on exposed sediments along the dewatered shoreline coupled with boat-generated turbulence could release more phosphorus than would normally occur if the lake was full of water. Under the right weather conditions, the disturbed sediments associated with implementation of Alternatives 2 or 3 could lead to more algae growth and potentially more risk to the workers involved in implementing either of these alternatives. The potential of exacerbated algae growth, over and above that predicted under Alternative 1, is expected to be limited however because the phosphorus available for plant growth (i.e. dissolved in the water column) has a strong tendency to bind to sediment particles. Its residence time as dissolved phosphorus in the water column would depend on the concentration of dissolved oxygen and pH in the lake water at the time. However, rapid uptake by sediment particles is expected (Johnson, 2004) with only a limited possibility of enhanced algae population growth under Alternative 2 and 3.

Because scores of people would be involved in the implementation of either Alternatives 2 or 3, the potential of administrative or application worker exposure to algae toxins is higher than the risk to administrative personnel only charged with water monitoring under Alternative 1. This is simply because more people would be at risk of accidental exposure to algae toxins under Alternatives 2 and 3 compared to Alternative 1 over the short-term.

Alternative 4 is expected to result in direct short-term public health hazards associated with exposure to toxic algae similar to that disclosed under Alternative 1. This is because Alternative 4 is expected to take about six years to affect tui chub populations, the primary driver of the toxic algae blooms. During this time frame, the people who visit and live at Diamond Lake would be susceptible to all the exposure pathways to anatoxin-a and microcystins described under Alternative 1. Alternative 4 presents the most risk to the health of administrative personnel from blue-green algae toxins because of the intense labor involved in fish removal and the sheer number of workers that would have to venture out on the lake every September (when a risk exists of exposure to algae toxins) to run gill nets and conduct other mechanical fish removal activities.

Indirect Effects:

Indirect effects to human health are those that would occur downstream of Diamond Lake and effects that would occur in the future, over the long-term.

The consumption of water or dermal exposure of water downstream of Diamond Lake in Lake Creek could potentially put members of the public at risk of illness under all alternatives. This downstream, indirect effect is only possible during the same risk periods of high algal toxin concentrations experienced in Diamond Lake itself. The blue-green algae toxins in Lake Creek would only be those delivered from Diamond Lake. Since Lake Creek has turbulent flow that is not conducive to algae proliferation, no additional growth of algae or associated toxic releases are expected to originate in Lake Creek itself. The likelihood of anyone receiving a dose of blue-green algae toxins out of Lake Creek would be lessened by the heightened public awareness and lake closures that would be put into effect as necessary, under all alternatives. These potential downstream indirect effects would be sustained over long periods of time under Alternatives 1 and 4, but would be short-lived under Alternatives 2 and 3 since Alternative 2 and 3 both rapidly reduce the tui chub population.

The risk of any indirect effect from toxic algae to the health of Lake Creek users would progressively lessen in a downstream direction as tributary streams, with no connection to Diamond Lake, enter Lake Creek and dilute toxin concentrations. Likewise, the risks to human health from exposure to algae toxins in Lemolo Lake, located 12 miles downstream of Diamond Lake, is much less than that of Lake Creek. No indirect downstream effects to human health at Lemolo Lake are expected to occur under any of the alternatives. This large reservoir is fed by many tributary streams, including the North Umpqua River, that function to mix and dilute blue-green algae toxins below levels of concern. No testing for blue-green algae toxin has occurred in Lemolo Lake.

Under Alternative 1, the indirect effects to human health over long periods of time are expected to be similar to the direct effects to human health disclosed above; the potential of serious illness or even death from toxic algae would be present through the same exposure pathways. However, the chances of actually experiencing impacts to human health associated with Alternative 1 increase with time simply due to the additive effects of summer after summer of potential public and administrative worker exposure. As time passes, assuming a fairly static population of tui chub, weather conditions could align to create toxic concentrations above those experienced to date.

On the other hand, cooler more overcast summers would result in fewer blooms and lower toxic concentrations. The threat of realizing indirect effects to human health over the long-term, under Alternative 1, can be expected to gradually increase over the years if the regional weather warming patterns continue on the same trajectory as the last few decades.

No long-term impacts to human health from exposure to toxic algae blooms are expected under either Alternative 2 or 3. Instead, Alternatives 2 and 3 are expected to result in indirect beneficial effects to human health hazard levels in the long-run relative to the existing condition. Predicting the effect of tui chub removal on the magnitude and annual trends of toxic algal blooms in Diamond Lake is difficult due to uncertainty in such factors as inter-annual climatic variability, restocking of the lake with rainbow trout, internal nutrient recycling from the sediments, and response of the zooplankton and benthic communities. Nonetheless, it is clear that based upon paleolimnological data that the lake began changing shortly after being stocked with trout, and that the greatest increases in *Anabaena* were associated with increased populations of tui chub (Eilers et al, 2001). Both Alternative 2 and 3 are therefore expected to result in a long-term lessening of risks to human health relative to Alternative 1, provided re-stocking with trout remains at a level that does not provide the same trophic and nutrient effect as the chubs.

Under Alternative 4, the indirect effects to human health over long periods of time are expected to be similar to the indirect effects to human health disclosed above for Alternative 1; the potential of serious illness or even death from toxic algae would be present through the same exposure pathways. However, under Alternative 4, the gradual long-term decline of tui chub can be expected to result in fewer toxic algae blooms, lessening public health risks proportionately. Table x summarizes the risks to the various groups of people if exposed to toxic algae in Diamond Lake.

Table 2. Summary of Exposure Risks to the Algal Toxins Associated with the Diamond Lake Restoration Project.

Alternative	Administrative or Application Workers	General visiting public and other residents
1	4 workers/summer at high risk of accidental exposure to algae toxins (during water sampling) over multiple summers for the foreseeable future.	Hundreds of water recreationists (swimmers, boaters, water skiers, sailboarders) could be exposed to algae toxins every summer if monitoring does not adequately detect toxin levels and lake closures are not timely.
2 & 3	25 workers at high risk of accidental exposure to algae toxins (during applications of rotenone from boats) and fish toxicants (while handling the concentrated formulations) during 1 summer.	No exposure anticipated.

Alternative	Administrative or Application Workers	General visiting public and other residents
4	15 workers/summer at high risk of accidental exposure to algae toxins during both water monitoring and fish removal work over multiple summers for the foreseeable future.	Hundreds of water recreationists (swimmers, boaters, water skiers, sailboarders) could be exposed to algae toxins every summer if monitoring does not adequately detect toxin levels and lake closures are not timely.

Cumulative Effects:

Under Alternative 1, the potential exists for cumulative effects to human health over years of chronic exposure to algal toxins. Uncertainty exists as to accumulation of anatoxin-a in fish tissue, so that fishermen may be at risk in eating their catches (Falconer 1993). Although it does not appear to be the case that recurrent low exposure to anatoxin-a leads to health problems later on, this is not established, and there is concern that people so exposed may become sensitized and develop increasingly more severe reactions with each new exposure (Backer 2002).

Anecdotal evidence for chronic effects of microcystins on humans is based in large part on the high rates of primary liver cancer in the rural regions of China where drinking water was obtained from ditches and ponds with large cyanobacteria loads. Where drinking water supplies have been changed from surface sources to deep wells, cancer rates have begun to drop (although other contributing factors are also being addressed). Other studies have shown microcystins to promote liver tumor development in lab animals. Yet, there is much uncertainty regarding the potential cumulative effects to human health under Alternative 1.

Under Alternatives 2 and 3, the possibility exists that workers exposed to both rotenone and algae toxins, while implementing either of these alternatives, could become more ill or more sensitized to the toxic effects of either of these toxins as a result of exposure to both. This potential cumulative effect would apply to the workers who would be implementing these alternatives, and the risk would be over the course of about a month. No studies have been done to confirm this possibility. Over the longer-term, within a few years of implementation, both Alternatives 2 and 3 would result in a beneficial effect to human health, lessening the possibility of chronic cumulative effects of toxic algae blooms compared to Alternative 1.

The potential cumulative effects to human health under Alternative 4 are similar to that of Alternative 1. Given the uncertainty of the long-term effectiveness of Alternative 4 at reducing the chub population and the fact that this alternative puts more workers at risk of exposure to algae toxins, the chances of cumulative effects to human health may actually be higher than Alternative 1. This is especially true if the same workers were repeatedly exposed year after year to the algal toxins.

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